

Canine pulmonary hypertension

Part 2: Diagnosis and treatment

Once a death sentence, canine pulmonary hypertension no longer carries such a grave prognosis—in part, because of advanced diagnostic tools and the availability of sildenafil therapy.

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Pulmonary hypertension is now being diagnosed in dogs relatively frequently.¹ To help you manage this once uncommonly diagnosed disease, in this article we review how to diagnose and treat canine pulmonary hypertension—and what the prognosis is for dogs with this condition.

To better understand this complex syndrome, be sure to read the first article in this series, “Canine pulmonary hypertension: An in-depth review of pathophysiology and classification.”

DIAGNOSIS

There are several goals in diagnosing pulmonary hypertension. The first is to identify the underlying cause or causes of pulmonary hypertension and to assign a clinical classification to the

patient. The second is to quantify the degree of pulmonary hypertension based on clinical signs and diagnostic test results. The third is to assess for hemodynamic impairment. And the fourth is to decide on the best treatment option for the patient.

Signalment and history

Most dogs with pulmonary hypertension are small-breed and middle-aged to older, which coincides with the high prevalence of pulmonary hypertension in patients with degenerative mitral valve disease.² Clients most often complain of exercise intolerance, as well as cough, dyspnea, and syncope.³ These signs occur because of pulmonary hypertension-induced impaired oxygen transport, reduced cardiac output, and systemic hypotension, resulting from systemic vasodilation and under filling of the right and left ventricles. Signs may also occur as a result of the underlying cause of pulmonary hypertension, such as interstitial lung disease.

Physical examination findings

Physical examination findings can include a heart murmur or a split S2 sound; the S2 heart sound is normally associated with the closure of the aortic and pulmonary valve leaflets. Abnormal lung sounds, ascites, and cyanosis have also been reported.²⁻⁵

Clinical pathology results

Given the vast assortment of underlying conditions that may lead to pulmonary hypertension, a thorough laboratory evaluation is recommended. A complete blood count, serum chemistry profile, heartworm antigen test, and urinalysis should be reviewed to help evaluate for systemic diseases that may predispose dogs to pulmonary hypertension. If indicated based on clinical signs, physical examination results, and baseline laboratory test results, patients may benefit from coagulation profiles, D-dimer concentrations, and endocrine testing.

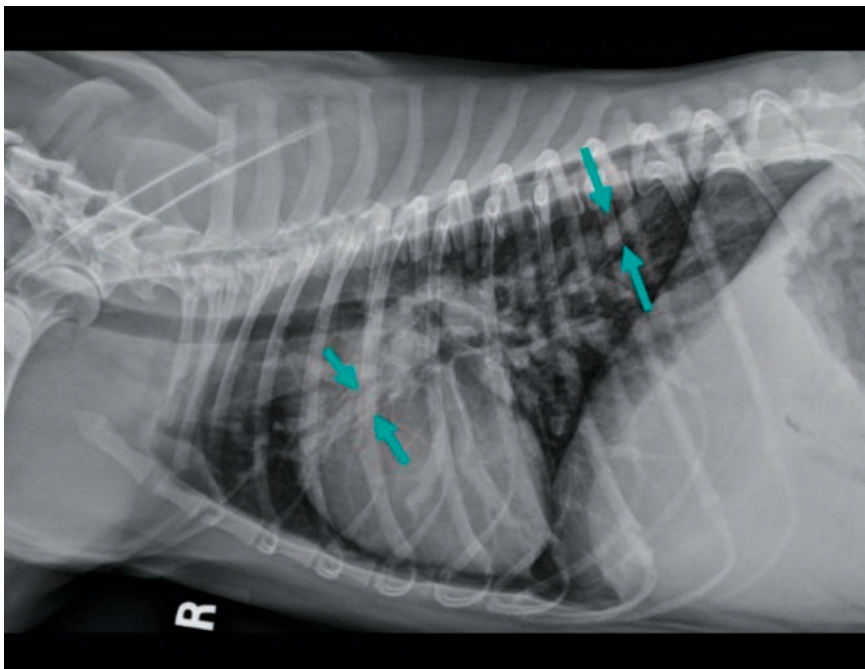
Biomarkers

Biomarkers such as brain natriuretic peptide (BNP) and cardiac troponins may be of added value in diagnosing patients with pulmonary hypertension.

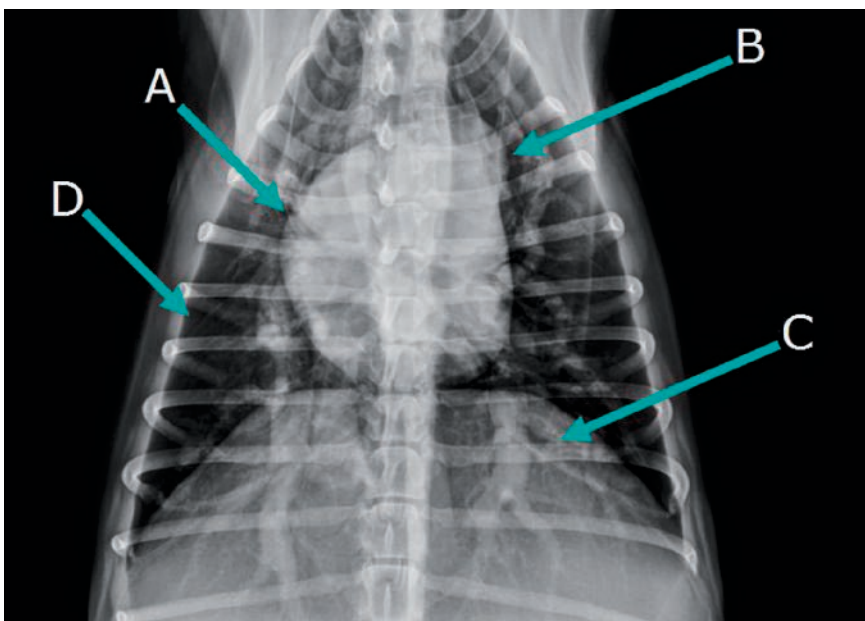
BNP is a hormone that is released from the ventricular myocardium in response to stress or strain.⁶ Although BNP is cleared quickly from circulation and is difficult to measure, cleavage of BNP produces a fragment—NT-proBNP—that remains in circulation longer and can be successfully measured. Traditionally, NT-proBNP measurements have been used in veterinary patients as a noninvasive means of determining the underlying cause of respiratory distress. More recent studies have shown that

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1A. A right lateral thoracic radiograph of a dog with clinical class I pulmonary hypertension secondary to heartworm disease. The cardiac silhouette is increased in size (the right ventricle appears especially prominent), and the pulmonary vessels are distended and tortuous (arrows). (Image courtesy of Ben Young, DVM, DACVR, Texas A&M University's Veterinary Medicine and Biomedical Sciences Department of Radiology.)



1B. A dorsoventral thoracic radiograph of a dog with clinical class I pulmonary hypertension secondary to heartworm disease. Arrow A indicates right-sided heart enlargement, arrow B demonstrates a prominent pulmonary artery, arrow C shows distended pulmonary vessels, and arrow D points to a fine pleural fissure line. (Image courtesy of Ben Young, DVM, DACVR, Texas A&M University's Veterinary Medicine and Biomedical Sciences Department of Radiology.)

NT-proBNP values are higher in dogs with clinical class III pulmonary hypertension than in patients with respiratory disease without associated pulmonary hypertension, those with moderate to severe pulmonary hypertension as based on pulmonary arterial pressure, or those with clinical class I, III, IV, or V pulmonary hypertension.^{2,7} In people with pulmonary hypertension, NT-proBNP values correlate with survival and can help predict the patient's prognosis.⁸

Cardiac troponins are proteins released from the ventricular myocardium as a result of myocardial cell injury and necrosis.⁹ Cardiac troponin I concentrations are commonly increased in dogs with clinical class I, II, III, or IV pulmonary hypertension.¹⁰

Thoracic radiography

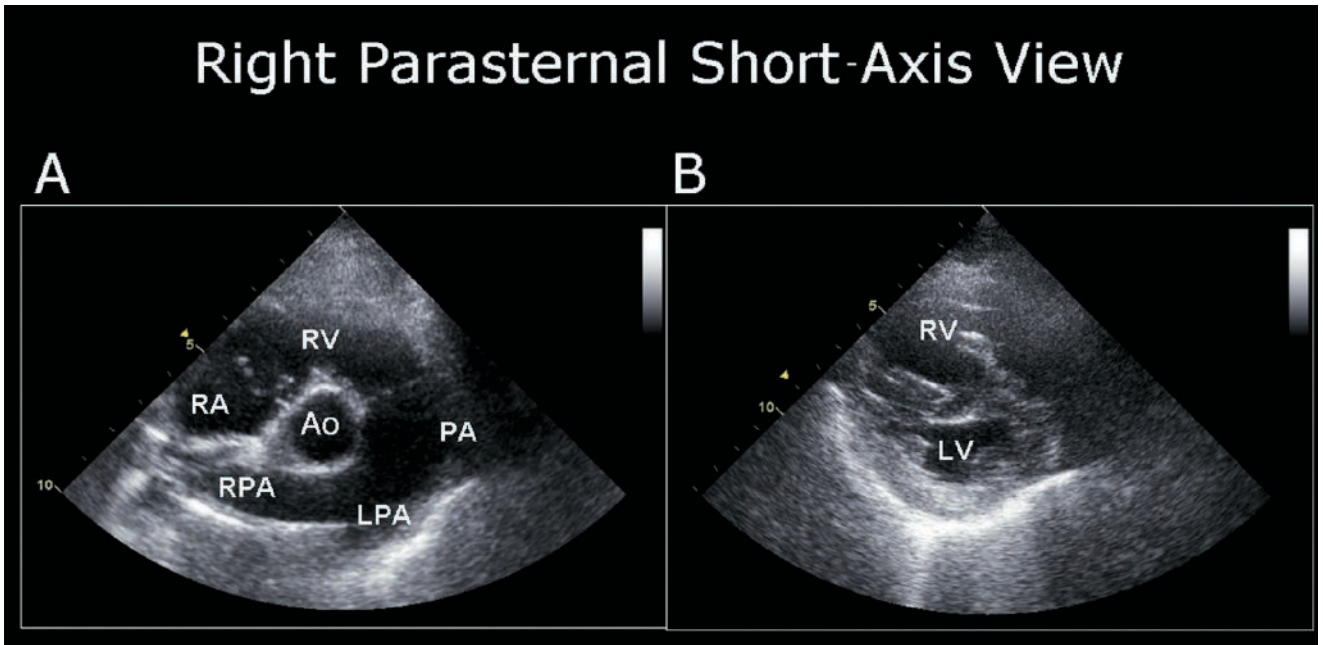
Thoracic radiography is not specific for pulmonary hypertension but may demonstrate findings supportive of it. Depending on the underlying cause of pulmonary hypertension, cardiomegaly, right-sided heart enlargement, or pulmonary artery dilation may be evident. In cases of congestive heart failure, pneumonia, or neoplasia, pulmonary infiltrates may be present. Pulmonary arteries may be tortuous in patients with heartworm disease (Figures 1A & 1B).

Electrocardiography

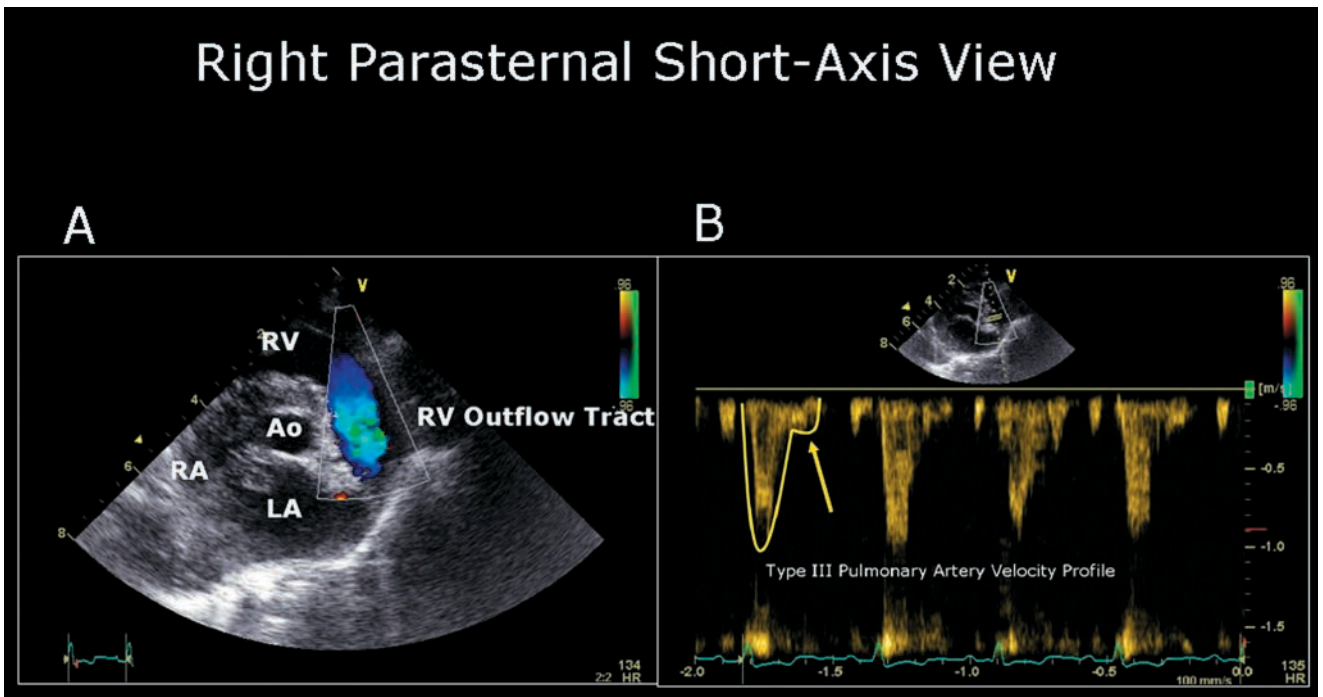
The electrocardiogram (ECG) is a non-specific test for pulmonary hypertension with low sensitivity. The ECG may be normal or show right axis deviation or a right-sided heart enlargement pattern.

Echocardiography

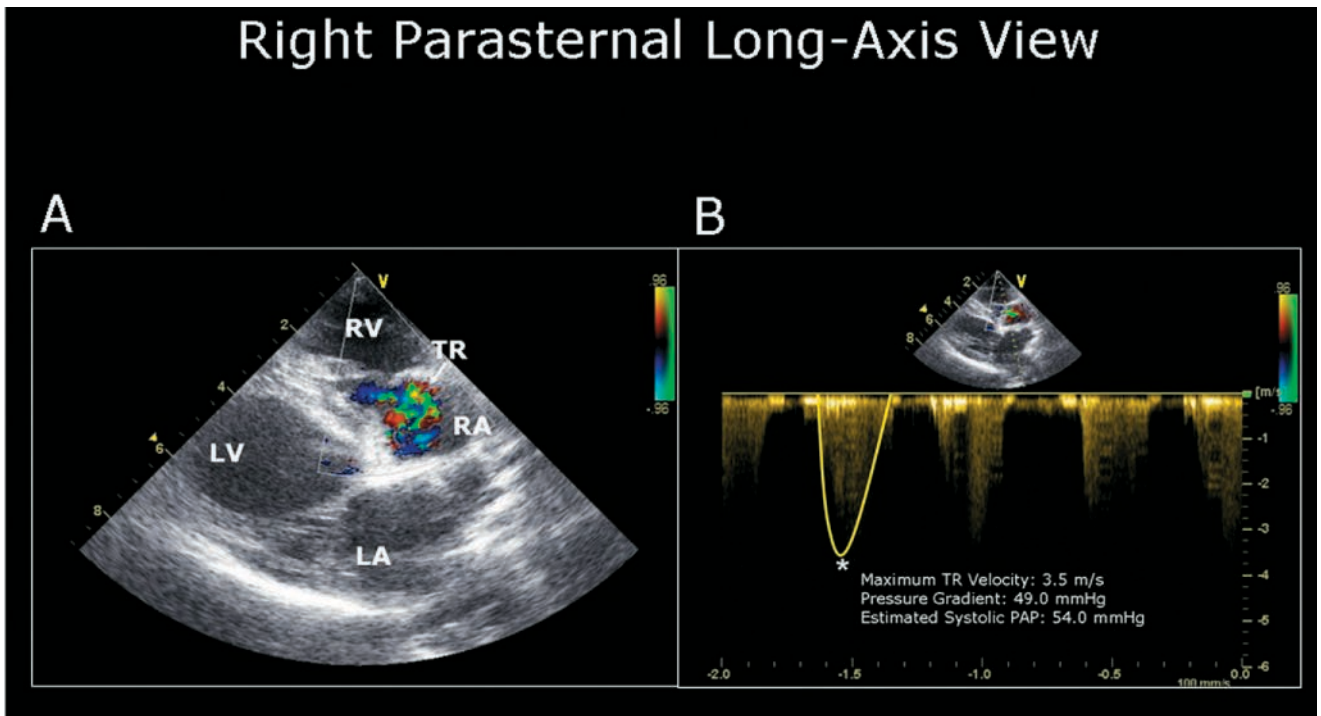
The echocardiogram is the gold standard noninvasive diagnostic tool used in veterinary medicine to diagnose pulmonary hypertension. Although tricuspid valve regurgitation maximum velocity measurement is the most common echocardiographic method used to diagnose systolic pulmonary hypertension in a clinical setting, other subjective and objective assessments are also possible. Subjective assessment of the cardiac



2. Right parasternal short-axis echocardiographic views of a dog with pulmonary hypertension. Panel A shows the basilar view of the heart, with moderate to severe pulmonary arterial dilation. Panel B shows the apical view of the heart, with severe septal flattening and moderate to severe right ventricular dilation (Key: Ao = aorta, RA = right atrium, RV = right ventricle, PA = pulmonary artery, RPA = right pulmonary artery, LPA = left pulmonary artery, LV = left ventricle).



3. A right parasternal short-axis echocardiographic view of a dog with pulmonary hypertension. Panel A shows color-flow Doppler across the pulmonary valve and blood flow across the valve. Panel B shows spectral Doppler measuring the velocity of the blood flow across the pulmonary valve and creating a pulmonary artery velocity profile. The first profile has been highlighted in yellow, with an arrow indicating the midsystolic notch. The pulmonary artery velocity profile is type III and supports a diagnosis of severe pulmonary hypertension (Key: LA = left atrium, RA = right atrium, RV = right ventricle, Ao = aorta).



4. A right parasternal long-axis echocardiographic view of a dog with pulmonary hypertension. Panel A shows color-flow Doppler of the tricuspid regurgitation jet. Panel B shows spectral Doppler measuring the velocity of the regurgitation, creating a tricuspid regurgitation velocity profile. The second tricuspid regurgitation velocity profile has been highlighted in yellow, and a star (*) approximates a maximum velocity of 3.5 m/s. When applied to the modified Bernoulli equation (pressure gradient = maximum velocity² x 4) a pressure gradient of 49 mm Hg is obtained. Given the mild right atrial dilation noted on echocardiogram, an estimated right atrial pressure of 5 mm Hg is added to the pressure gradient, estimating systolic pulmonary artery pressure at 54 mm Hg. This patient has moderate systolic pulmonary arterial hypertension (Key: LA = left atrium, LV = left ventricle, RV = right ventricle, TR = tricuspid regurgitation, RA = right atrium).

structures and pulmonary artery velocity profiles can aid in diagnosing pulmonary hypertension, and objective assessment of pulmonary valve insufficiency can help diagnose and quantify diastolic pulmonary hypertension.

Subjective echocardiographic findings. Patients with pulmonary hypertension have increased right ventricular afterload and may develop right-sided heart changes associated with this pressure increase. Right ventricular and atrial chamber sizes may be normal or mildly to severely dilated. Right wall thicknesses may be normal, or some degree of right ventricular hypertrophy may be evident (Figure 2). Paradoxical septal motion and septal flattening support right ventricular pressure overload. Pulmonary artery dilation is often seen in patients with pulmonary hypertension, and in cases of heartworm infection, heartworms

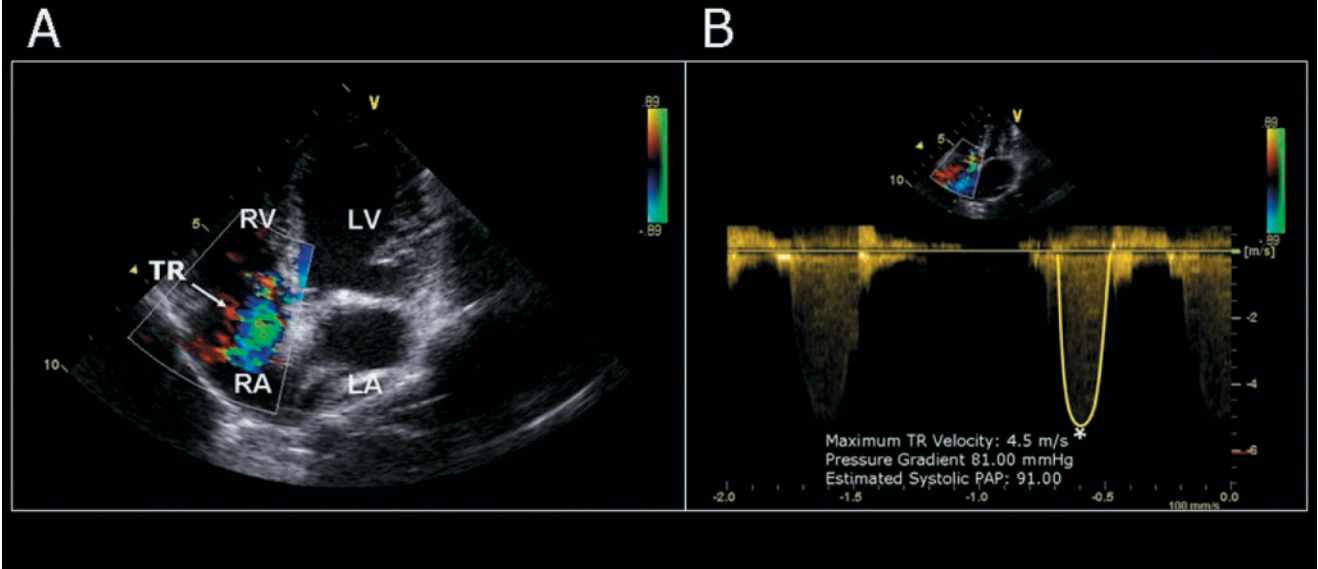
may be visualized in the right heart or right pulmonary artery.

Pulmonary artery velocity profiles may also aid in the echocardiographic diagnosis of pulmonary hypertension. Spectral Doppler echocardiography is used to measure the outflow velocity across the pulmonary valve by generating a pulmonary artery velocity profile.¹⁰ In patients with normal pulmonary arterial pressure, a type I profile is recorded with equal acceleration and deceleration times producing a symmetrical appearance. As pulmonary arterial pressures increase, an asymmetrical profile is recorded because of early rapid acceleration and an earlier peak velocity. This asymmetrical profile occurs as a result of increased rate of rise in pulmonary vascular pressures and is referred to as a *type II profile*. Type II profiles are associated with mild to moderate pulmonary hypertension.

With severe pulmonary hypertension, a rapid acceleration with an earlier peak velocity occurs, and a midsystolic notch is also present. This is referred to as a *type III profile*. The midsystolic notch indicates blood flow reversal secondary to high pulmonary vascular pressures (Figure 3).

Doppler assessment of systolic pulmonary arterial pressure. Tricuspid valve regurgitation occurs during ventricular systole and can be viewed during an echocardiogram in right parasternal and left apical views (Figures 4 & 5). In these views, the tricuspid regurgitation jet is assessed by using color-flow Doppler, and spectral Doppler is placed over the tricuspid regurgitation jet to obtain tricuspid regurgitation velocity profiles. The peak of the profile indicates maximum tricuspid regurgitation velocity.

Left Apical View



5. A left apical echocardiographic view of a dog with pulmonary hypertension. Panel A shows color-flow Doppler of the tricuspid regurgitation jet. Panel B shows spectral Doppler measuring the velocity of the regurgitation, creating a tricuspid regurgitation velocity profile. The second tricuspid regurgitation velocity profile has been highlighted in yellow, and a star (*) approximates a maximum velocity of 4.5 m/s and, when applied to the modified Bernoulli equation, a pressure gradient of 81 mm Hg is obtained. Given the moderate right atrial dilation noted on echocardiogram, an estimated right atrial pressure of 10 mm Hg is added to the pressure gradient, estimating systolic pulmonary artery pressure at 91 mm Hg. This patient has severe systolic pulmonary arterial hypertension (Key: RA = right atrium, TR = tricuspid regurgitation, RV = right ventricle, LV = left ventricle, LA = left atrium).

The maximum tricuspid regurgitation velocity estimates the pressure gradient across the tricuspid valve, which approximates the pressure difference between the right ventricle and right atrium. The pressure gradient can be calculated by applying the measured maximum tricuspid regurgitation velocity to the modified Bernoulli equation³:

$$\text{pressure gradient} = \text{maximum tricuspid regurgitation velocity}^2 \times 4$$

When an estimated right atrial pressure is added to the calculated pressure gradient, an estimate of right ventricular pressure can be obtained. Right atrial pressures are estimated based on subjective assessment of the right atrium's size on an echocardiogram. A normal right atrium is assigned a pressure of 0 mm Hg, a mildly dilated right atrium

has an estimated pressure of 5 mm Hg, a moderately dilated right atrium has an estimated pressure of 10 mm Hg,

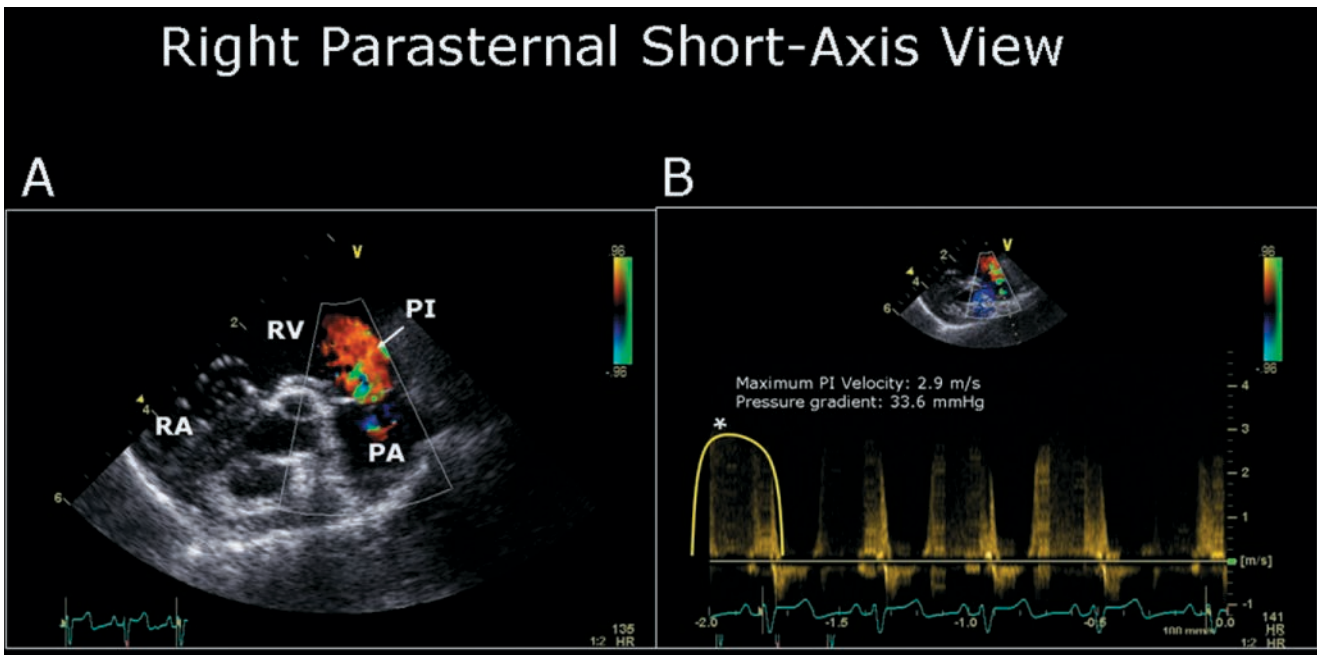
pressure and, thus, can help identify and stratify pulmonary hypertension. Normal systolic pulmonary arterial

Diastolic pulmonary arterial pressure > 15 mm Hg supports a diagnosis of diastolic pulmonary arterial hypertension.

and a severely dilated right atrium has an estimated pressure of 15 mm Hg.^{11,12}

In the absence of a right ventricular outflow tract obstruction, the right ventricular systolic pressure is an estimate of systolic pulmonary arterial

pressure is < 25 mm Hg, mild systolic pulmonary arterial hypertension is 30 to 50 mm Hg, moderate systolic pulmonary arterial pressure is 50 to 80 mm Hg, and severe systolic pulmonary arterial pressure is > 80 mm Hg.¹³



6. A right parasternal short-axis echocardiographic view of a dog with pulmonary hypertension. Panel A shows color-flow Doppler of the pulmonary insufficiency jet. Panel B shows spectral Doppler measuring the velocity of the insufficiency, creating a pulmonary insufficiency velocity profile. The first pulmonary insufficiency velocity profile has been highlighted in yellow, and a star (*) approximates a maximum velocity of 2.9 m/s and, when applied to the modified Bernoulli equation, a pressure gradient of 33.6 mm Hg is obtained. Assuming the right ventricular pressure in diastole is 0 mm Hg, the estimated diastolic pulmonary arterial pressure is 33.6 mm Hg. This patient has diastolic pulmonary arterial hypertension (Key: RA = right atrium, RV = right ventricle, PI = pulmonary insufficiency, PA = pulmonary artery).

Doppler assessment of diastolic pulmonary arterial pressure. Pulmonary valve insufficiency occurs in diastole and can be viewed best through the right parasternal short-axis view (Figure 6). In this view, the pulmonary insufficiency jet is assessed by using color-flow Doppler, and spectral Doppler is placed over the pulmonary insufficiency jet to obtain pulmonary insufficiency velocity profiles. The peak of the profile indicates maximum pulmonary insufficiency velocity (Figure 6, panel B).

The maximum pulmonary insufficiency velocity estimates the diastolic pressure gradient across the pulmonary valve, or the pressure difference between the pulmonary artery and right ventricle. The pressure gradient can be calculated by applying the measured maximum pulmonary insufficiency velocity to the modified Bernoulli equation. The right ventricular pressure in diastole is assumed to be 0 mm Hg. Thus, the pressure gradient across the pulmonary valve in diastole equals the diastolic

pulmonary arterial pressure. Normal diastolic pulmonary arterial pressure is < 15 mm Hg, and any value higher than that supports a diagnosis of diastolic pulmonary arterial hypertension.¹³

Echocardiographic errors and limitations. Although subjective analysis of the right ventricle, pulmonary artery, and septal wall motion can be helpful in diagnosing pulmonary hypertension, patients with pulmonary hypertension may not always demonstrate these echocardiographic findings. Additionally, identifying heartworms on an echocardiogram can be difficult, and echo artifacts may lead to a false positive diagnosis.

The assessment of the pulmonary artery velocity profile types is not always accurate, and veterinary and human studies suggest that up to 50% of patients with documented pulmonary hypertension may not have pulmonary artery velocity profile changes consistent with pulmonary hypertension.¹³ How-

ever, one study in dogs found that the short acceleration times and earlier peak velocities associated with type II and III pulmonary artery velocity profiles have 80% sensitivity and specificity for diagnosing pulmonary hypertension.¹⁴

In patients with right ventricular outflow tract obstruction, such as in pulmonic stenosis, tricuspid regurgitation, and pulmonary insufficiency, maximum velocity measurements are not accurate estimates of pulmonary arterial pressure and cannot be used to diagnose pulmonary hypertension. Human literature cites that in patients with tricuspid regurgitation or pulmonary insufficiency who are otherwise normal, maximum velocity measurements may overestimate or underestimate pulmonary arterial pressure by 20 mm Hg.¹³

Pulmonary arterial pressure can be overestimated if regurgitation jets overlap and color-flow and spectral Doppler placement is incorrect. In human medicine, there is evidence that adding estimated right atrial pressure

may overestimate systolic pulmonary arterial pressure.¹⁵ Pulmonary arterial pressure may be underestimated if there is poor Doppler signal strength, poor jet alignment, or right ventricular myocardial failure or if tricuspid regurgitation and pulmonary insufficiency jets cannot be identified.^{15,16}

Other diagnostic tools

More advanced echocardiographic techniques can be used to diagnose pulmonary hypertension but are not commonly used. These techniques include calculating a Tei Index, which assesses myocardial function.^{2,14} Tissue Doppler and right ventricular systolic time intervals can also be evaluated.¹⁴

In human medicine, right heart catheterization is the gold standard for di-

agnosing pulmonary hypertension.^{17,18} In veterinary patients, this procedure requires general anesthesia and is a high-risk procedure given most patients' clinical signs. Right heart catheterization is also expensive and not commonly used in the clinical veterinary setting to diagnose pulmonary hypertension. Computed tomographic angiography, pulmonary function testing, and ventilation perfusion scans are also used in people.¹⁹

TREATMENT

Treating canine pulmonary hypertension should be approached with several goals in mind. First and foremost, treatment should be aimed at decreasing clinical signs: improving exercise intolerance, dyspnea, and cough and decreasing syncopal events. In addition, treatment should also be instituted to decrease the patient's hospitalization time, improve

quality of life, and increase survival time. These goals can be accomplished by identifying and treating the underlying cause of pulmonary hypertension and, often, by using medications to help decrease pulmonary arterial pressure and the workload of the right ventricle. Based on recommendations from our human counterparts, therapy should be started in any patients with functional class III or IV disease, with the goal of reassigning them to class I or II.²⁰

Prostacyclin analogues

Prostacyclin is a known vasodilator and platelet inhibitor and also has antiproliferative effects.^{8,13} Prostacyclin analogues include epoprostenol, treprostinil, and iloprost. Epoprostenol and treprostinil are given intravenously. Treprostinil can

be given subcutaneously, and iloprost is given as an inhalant. In people, these medications improve symptoms and survival. Side effects include anemia, thrombocytopenia, hypotension, and gastrointestinal symptoms.⁸

Prostacyclin analogues have been found to be experimentally effective in treating canine pulmonary hypertension, but there are no published clinical trials. Prostacyclin analogues improve cardiac output, vasodilation, and right ventricular performance.^{21,22} Unfortunately, prostacyclin analogues are exorbitantly expensive and are not practical for administration in veterinary patients.

Endothelin antagonists

As discussed previously in Part 1 of this article series, endothelin-1 causes vasoconstriction, smooth muscle proliferation, and vascular remodeling. By antagonizing the actions of endothelin-1,

pulmonary arterial pressure can be decreased. The most common endothelin antagonists include bosentan, sitaxsentan, and ambrisentan. These oral medications have been shown to improve exercise intolerance, pulmonary arterial pressure, and even pulmonary vascular resistance in people.^{8,23} Side effects in people include dose-dependent hepatotoxicosis, anemia, and birth defects.⁸

Bosentan has been assessed in dogs in experimental settings. In these patients, bosentan was effective and was been found to decrease vascular remodeling in induced pulmonary hypertension, improve myocardial function, and decrease ventricular remodeling.^{24,25} Unfortunately, there are no clinical trials of endothelin antagonists in dogs with naturally occurring disease. Endothelin antagonists, while less expensive than prostacyclin analogues, are considered cost-prohibitive in veterinary patients.

Nitric oxide and nitrates

Nitric oxide is an inhaled vasodilator, while isosorbide dinitrate and isosorbide mononitrate are oral vasodilators. Nitrates are given to people for their antianginal effects but have limited application for treating pulmonary hypertension. These medications are not commonly used in veterinary medicine because of potentially severe side effects.

L-arginine, the precursor to nitric oxide, has been administered to people with pulmonary hypertension. This drug is given orally and may improve nitric oxide concentrations and cause vasodilation. In people, this drug is questionably effective, but there are no studies in dogs.²⁶

Phosphodiesterase 5 inhibitors

There are several phosphodiesterase 5 (PDE5) inhibitors, including sildenafil (Viagra—Pfizer), tadalafil (Cialis, Adcirca—Eli Lilly), and vardenafil (Levitra, Staxyn—GlaxoSmithKline). These drugs all specifically inhibit PDE5, which is highly concentrated in pulmonary vessels. As discussed in the Part 1 of this article series, PDE5 normally breaks down cyclic guanosine monophosphate

Sildenafil is considered the drug of choice for pulmonary hypertension in dogs.

(cGMP). When PDE5 is inhibited, cGMP concentrations increase and vasodilation is promoted. In people, PDE5 inhibitors all cause pulmonary arterial vasodilation, but there are subtle differences between the three substances in regard to onset and duration of action, degree of decreased pulmonary vascular resistance, and ability to reverse cardiac hypertrophy.^{8,27}

Sildenafil is a short-acting PDE5 inhibitor. It has been evaluated for treating canine pulmonary hypertension in the clinical setting and has been shown to decrease pulmonary arterial pressure, improve quality of life, and improve survival time.^{28,29} Sildenafil has rare gastrointestinal side effects and, while relatively expensive, may be affordable for many clients. It is considered the drug of choice for treating pulmonary hypertension in dogs. The recommended sildenafil dose is 1 to 3 mg/kg by mouth every eight to 12 hours.

Tadalafil is a long-acting PDE5 inhibitor. There is a single published case study evaluating a dog treated with tadalafil. The patient was diagnosed with idiopathic pulmonary hypertension and was treated with tadalafil (1 mg/kg orally every 48 hours) in addition to other medications.³⁰ The patient demonstrated decreased pulmonary arterial pressure and improved clinical signs with tadalafil treatment but was euthanized 10 days later because of signs of weakness, tremor, and decreased appetite.³⁰ Ultimately, the clinicians felt the patient suffered systemic hypotension secondary to tadalafil use, but that was not confirmed. While there is some evidence that tadalafil can improve echocardiographic signs and clinical signs in dogs with pulmonary hypertension, this medication should be used cautiously.

Vardenafil is another long-acting PDE5 inhibitor. To date, vardenafil has not been studied either experimentally or in the clinical setting for its effectiveness in treating canine pulmonary hypertension and should not be considered if other options are available.

Other phosphodiesterase inhibitors

Theophylline is a weak, nonselective PDE inhibitor and affects PDE3, PDE4, and PDE5. This medication is used commonly for its ability to relax smooth muscle in patients with bronchial disease. In human medicine, it has been shown to improve pulmonary hypertension secondary to chronic obstructive pulmonary disease and may be useful in dogs with clinical class III pulmonary hypertension. In general, theophylline is not considered an effective single agent for treating pulmonary hypertension.²

Pimobendan (Vetmedin—Boehringer Ingelheim Vet-medica) and levosimendan are dual mechanism drugs. They exert positive inotropic effects associated with calcium sensitization as well as vasodilatory effects mediated by PDE3 inhibition. Clinically, pimobendan has been shown

to improve pulmonary hypertension secondary to degenerative mitral valve disease (clinical class II).³¹ Pimobendan has not been well-studied as a single agent therapy in dogs with other clinical classes of pulmonary hypertension and is not considered an effective therapy for those dogs with pulmonary hypertension for any clinical class other than II.²

Supportive care and treatment of underlying conditions

Patients with pulmonary hypertension often require supportive therapy and additional therapies based on the underlying cause of the pulmonary hypertension. These therapies may include oxygen supplementation, treatment of pulmonary disease with anti-inflammatory or bronchodilating agents, treatment of neoplasia with appropriate chemotherapy, or treatment of congestive heart failure with diuretics. Patients with thromboembolic disease may benefit from anticoagulant medications and corticosteroids, and patients with heartworm disease may require heartworm adulticide therapy.

Monitoring therapy

Once therapy is instituted, monitoring is most often based on the patient's clinical signs. Sildenafil therapy may be increased or decreased within the dose range according to clinical signs, but the World Health Organization's Evian Functional Classification



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scheme (see Table 2 in the previous article) more objectively assesses the therapeutic response. For instance, a patient may initially be categorized as functional class IV, but after four weeks of therapy

can estimate pulmonary arterial pressures and assess the severity of pulmonary hypertension. Historically, the prognosis for dogs with pulmonary hypertension was grave. However, with the advent of

Before the availability of sildenafil, dogs with pulmonary hypertension had grave prognoses.

be reevaluated as functional class II.

Recheck thoracic radiography may show improvement in pulmonary infiltrates and right-sided cardiac enlargement. A repeat echocardiographic examination may show partial or complete resolution of right-sided heart changes and improved pulmonary artery velocity flow profiles. Evidence of decreased pulmonary arterial pressure may be noted in the form of decreased maximum tricuspid regurgitation or pulmonary insufficiency velocities.

PROGNOSIS

The prognosis for dogs with pulmonary hypertension varies and often depends on the underlying cause. Before the availability of sildenafil, dogs with pulmonary hypertension had grave prognoses, with many surviving only days past diagnosis.³ With the introduction of sildenafil therapy, the average published survival has increased to 91 days, with some patients surviving almost two years.²⁸ Negative prognostic factors may include advanced functional class, high right atrial pressure, severe right ventricular dysfunction, and increased NT-proBNP values.^{14,29}

CONCLUSION

Although several diagnostic tools may be used to help diagnose canine pulmonary hypertension, an echocardiographic examination is the gold standard in veterinary medicine. By using echocardiography, you

can estimate pulmonary arterial pressures and assess the severity of pulmonary hypertension. Historically, the prognosis for dogs with pulmonary hypertension was grave. However, with the advent of advanced diagnostic tools, the ability to recognize and treat underlying diseases, and the availability of sildenafil, prognosis has improved significantly. ❖

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